Mechanism of failure in the treatment of type II endoleak with percutaneous coil embolization

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Purpose: **Type II endoleak after endovascular abdominal aortic aneurysm repair is a failure of aneurysm sac exclusion with unknown long-term consequences. Elevated aneurysm sac pressures documented in these patients have led us to aggressively treat type II endoleaks with percutaneous transluminal coil embolization (PTCE). The purpose of this study was to evaluate the results and the mechanisms of failure of PTCE for type II endoleak.**

Methods: **One hundred ninety-one patients underwent endograft repair of infrarenal aortic aneurysms. Twenty-three of 28 patients with persistent primary (>3 months) or secondary (new-onset) endoleak underwent angiography; 14 of these patients had type II endoleaks. We reviewed our endovascular registry data, hospital charts, and radiologic studies of patients with type II endoleaks and analyzed the results in those treated with PTCE of the inflow vessel.**

Results: **All 14 patients with type II endoleaks were men, with a mean age of 76.7 years and a mean preoperative maximal aneurysm diameter of 5.7 ± 1.0 cm. The type II endoleak was primary in 12 patients (86%) and secondary in two patients (14%) and iliolumbar in 11 patients (78%) and mesenteric in three patients (21%). Although a dominant affluent collateral channel (inosculation) was apparent in eight patients (57%), six patients (43%) showed a network of collateral vessels (retiform anastomosis). In six patients (43%), angiography revealed a second or "outflow" vessel indicative of a complex endoleak. In four patients with retiform iliolumbar type II endoleaks, PTCE was not attempted because of the retiform nature of the endoleak. The remaining 10 patients underwent PTCE, with coil deployment in all 10 and apparent initial technical success in nine patients. Follow-up computed tomographic scans revealed persistent endoleaks in six patients (60%). Mechanisms of failure included persistent flow through the coils in the treated vessel in two patients, development of a retiform anastomosis around the coiled vessel in three patients, and development of a new mesenteric endoleak after successful occlusion of an iliolumbar endoleak in one patient. Two patients underwent repeat PTCE with successful aneurysm sac exclusion in one. Internal iliac artery injury complicated one of the 12 PTCEs, and the resulting pseudoaneurysm was successfully treated with PTCE. Angiographic visualization of an outflow vessel (complex endoleak) was associated with PTCE failure ($P = .008$).**

Conclusion: **PTCE of type II endoleaks has a high failure rate because of multiple anatomic mechanisms. (J Vasc Surg 2002;36:485-91.)**

A *type II endoleak* after endoluminal repair of an abdominal aortic aneurysm (er-AAA) refers to collateral blood flow retrograde into the sac from patent aortic branch vessels, usually the lumbar or the inferior mesenteric artery (IMA).[1] Although completion angiograms after endograft implantation commonly show one or more patent lumbar arteries or the IMA, most of these aortic side branches will thrombose in the early postoperative period. Continued patency of any branch vessel in continuity with the aneurysm sac is defined as a *type II endoleak*.

Because of the limited follow-up available, the management of type II endoleaks after er-AAA remains controversial. Nonetheless, a type II endoleak clearly indicates a failure of aneurysm sac exclusion, and both clinical and laboratory investigations have documented elevated sac pressures.[2,3] Moreover, continued aneurysm enlargement and post–er-AAA rupture have been reported in patients with persistent type II endoleaks.[4-6] This evidence has led our group to follow an aggressive management algorithm of early angiography and percutaneous transluminal coil embolization (PTCE) of type II endoleaks. Herein, we present our results of PTCE and analyze the mechanisms of failure of PTCE for type II endoleak.

METHODS AND MATERIALS

Study population and design. Between June 1997 and December 2001, 191 patients underwent successful deployment of an aortic endograft at Southern Illinois University School of Medicine and Memorial Medical Center. The devices used included: AneuRx endograft system (Medtronic/AVE, Inc, Santa Rosa, Calif), Excluder (WL Gore and Associates, Inc, Flagstaff, Ariz), AneuRx AUI (Medtronic/AVE, Inc), Talent endoluminal spring stent graft (Medtronic, Inc, Sunrise, Fla), and Power Link System (Endologix, Inc, Irvine, Calif). Data from all patients undergoing er-AAA were entered prospectively into an endovascular registry. The surveillance protocol adopted at

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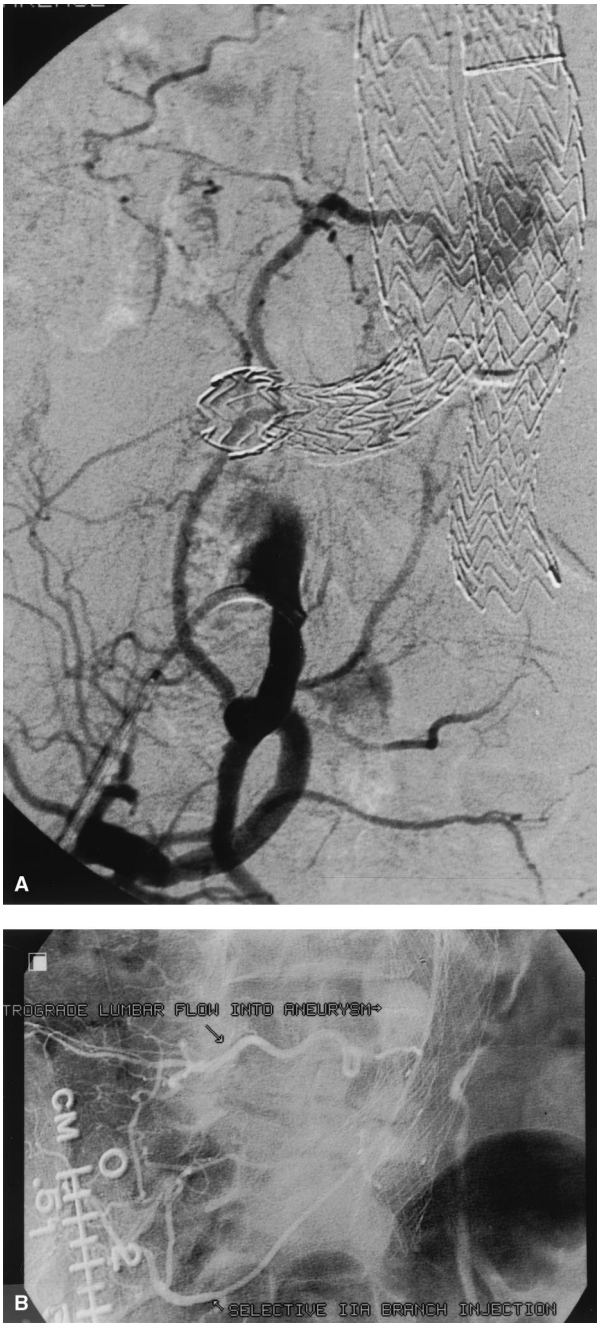

A, Selective internal iliac artery angiogram shows iliolumbar type II endoleak with single large collateral vessel (inosculation) filling aneurysm sac *(arrow)*. **B,** Selective internal iliac artery angiogram shows iliolumbar type II endoleak composed of network of small collateral vessels (retiform anastomosis).

Southern Illinois University after er-AAA has been previously reported.[7] All patients undergo computed tomographic (CT) scanning at 1 month. If the imaging at 1 month indicates the presence of an endoleak, a CT scan is performed at 3 months after repair. An angiogram is performed if an endoleak is still present at 3 months. If no endoleak is noted on the 1-month scan, or on the 3-month scan for those positive at 1 month, further follow-up CT scans are performed at 6 months and 1 year and annually thereafter. All patients with the development of secondary (new-onset) endoleaks undergo angiography, and more frequent CT scanning may be undertaken in patients who have persistent endoleaks, undergo secondary interventions, or show evidence of aneurysm enlargement.

Data from our endovascular registry, hospital records, and clinic charts were reviewed for all patients with type II endoleaks. CT scans and angiograms were reviewed by a single radiologist (GB) blinded to the treatments and outcomes. χ^2 and Student t tests were used to determine differences between groups. Significance was assigned a P value of .05 or less.

Classification of type II endoleak. A type II endoleak can be classified as primary or secondary. A *primary endoleak* is detected on the 1-month CT scan and is presumed to have been present from the completion of the endograft repair. A *secondary endoleak* develops after a period of complete sac exclusion as determined with follow-up imaging studies. Type II endoleaks can be further classified by the angiographic anatomy as iliolumbar, mesenteric, or other. An *iliolumbar type II endoleak* originates from the posterior trunk of the internal iliac artery that supplies, via the iliolumbar branch or unnamed collaterals, one or more ipsilateral lumbar arteries and the aneurysm sac. A *mesenteric type II endoleak* originates from the middle colic artery branch of the superior mesenteric artery that fills a patent IMA and the aneurysm sac via the arc of Riolan or the marginal artery, or more rarely the IMA may fill from the pelvis via the superior rectal arteries. Rarely, endoleaks can result from a patent accessory renal or middle sacral artery and may be classified as other. We also have found it useful to classify the character of the collateral pathway as a retiform anastomosis or an inosculation. *Retiform anastomosis* describes a collateral pathway composed of a network of small arteries. Collateral blood flow through a single relatively large dominant vessel is a phenomenon termed *inosculation* (Fig).[8] Finally, type II endoleaks may also be classified as simple or complex. *Simple endoleaks* have only one vessel in communication with the aneurysm sac, whereas *complex type II leaks* have multiple communicating vessels. Visualization of contrast exiting the aneurysm sac through outflow vessels during selective angiography is indicative of a complex type II endoleak.

Percutaneous transluminal coil embolization. Coil embolization of type II endoleaks was performed in an angiographic suite. Iliolumbar endoleaks were initially approached from the ipsilateral femoral artery; however, in two patients, stable catheter positions could not be achieved and a left brachial artery access site was used. Mesenteric endoleaks were approached from either femoral artery. In most cases, a 5F access sheath was used for diagnostic angiography and anticoagulation was achieved with 5000 U of heparin. If needed, the diagnostic sheath was upsized to 6F or 8F to accommodate guiding catheters

of various configurations, which were positioned in the proximal superior mesenteric artery or internal iliac artery. Superselective catheterization of the target ipsilateral lumbar artery or IMA was accomplished with a hydrophilic coated 0.014 Transcend wire (Boston Scientific, Medi-Tech, Boston, Mass) and a 3F Renegade fiber braided microcatheter (Boston Scientific Cork, Ltd, Cork, Ireland). Tornado embolization coils (Cook Group Company, Bloomington, Ind), 3 to 5 mm in maximal diameter, were used to embolize the aneurysm sac or the inflow vessel as close to the aneurysm sac as possible.

RESULTS

Of the 191 patients who underwent er-AAA during the study period, our surveillance protocol identified 28 patients (14.6%) with primary and secondary (new-onset) endoleaks. In four patients, primary endoleaks detected at 1 month spontaneously resolved by 3 months. An additional small secondary endoleak also resolved before angiography. Diagnostic angiography was performed in the remaining 23 patients and revealed type I endoleaks in two patients and type III endoleaks in three patients, with angiography indeterminate in four patients and type II endoleaks in 14 patients.

All 14 patients with type II endoleaks were men, with a mean age of 76 ± 8.19 years (range, 63 to 90 years). AAA characteristics and endografts used are given in Table I. The type II endoleaks were primary in 12 patients (86%) versus secondary in two patients (14%) and iliolumbar in 11 patients (78%) versus mesenteric in three patients (21%). Although a dominant collateral channel (inosculation) was apparent in eight patients (57%), six patients (43%) showed a retiform anastomosis (Fig). In six patients (43%), angiography revealed a second or outflow vessel indicative of a complex endoleak.

Four patients with small, retiform, iliolumbar type II endoleaks were not treated because catheterization of the inflow lumbar artery was not considered technically feasible because of the retiform characteristics of the collateral network. Spontaneous resolution of the endoleak occurred in one of the four patients. In the remaining three patients, aneurysm sac diameters have remained stable or decreased in size (mean diameter change, −3 mm). Two of the four untreated patients died in follow-up of causes unrelated to the aortic aneurysm.

Ten patients underwent PTCE, with coil deployment in all 10 and apparent initial technical success in nine patients (Table II). One patient had a technically inadequate PTCE with a single coil placed in a proximal internal iliac branch supplying a retiform endoleak. Follow-up CT scans revealed persistent endoleaks in six of the 10 treated patients. All six patients underwent repeat angiographic evaluation, which revealed multiple mechanisms of failure of PTCE to achieve aneurysm sac exclusion.

In one patient, angiography revealed successful obliteration of the treated iliolumbar endoleak but the development of a new mesenteric endoleak. This mesenteric endoleak, which may have contributed to the endoleak initially but was not evident on previous transfemoral angiograms, was successfully treated with repeat PTCE with complete resolution of all endoleak on follow-up CT scan.

Table I. Demographics, preoperative AAA characteristics, and endograft devices used in 14 patients with type II endoleaks

Patient and AAA characteristics	
Age (y; mean ± standard deviation)	76.3 ± 8
Preoperative AAA diameter (mm ± SD)	57 ± 9.9
No. of patent lumbar arteries	4.4 ± 1.1
Preoperative patency of IMA	8 (57%)
Type of endograft used	
AneuRx bifurcated	10 (71%)
AneuRx AUI	2 (14%)
Excluder	1 (7%)
Talent	1 (7%)

In two patients, angiography showed persistent blood flow through the coils of the treated endoleak vessels. In one case, continued patency of a thoroughly coiled IMA was seen in a patient who needed anticoagulation therapy for a prosthetic heart valve. At 24 months of follow-up, CT scanning has documented a stable aneurysm diameter in this patient with no further intervention. The patient with a technically inadequate initial PTCE had persistent flow through the single proximally deployed coil. Follow-up CT scans in this patient documented a 0.5-cm increase in aneurysm diameter that prompted a second PTCE with multiple 3-mm coils deployed distally in the endoleak channels. No further growth of the aneurysm sac has been noted despite persistence of the endoleak after 9 months of follow-up.

In the remaining three patients, angiography showed persistent iliolumbar endoleaks caused by the new development of a retiform anastomosis around the previously coiled vessel. None were considered amenable to further coil embolization. One of these three patients had a significant (0.7 cm) increase in aneurysm sac diameter, and translumbar sac embolization is planned.

Overall, PTCE was performed 12 times in 10 patients and was successful in thrombosis of the treated endoleak vessel in six (50%). No significant differences were found between successful and failed PTCE procedures in regards to patient age, type of endograft implanted, preoperative aneurysm diameter, number of lumbar arteries present, and IMA patency. No difference in outcomes was found between successful and failed PTCE with regard to the number of coils deployed (10.3 ± 4 versus 7.0 ± 4) or whether coils were placed into the aneurysm sac itself (2/4, 50% versus 2/6, 33%) nor with regard to any classification of the endoleak, whether primary versus secondary, iliolumbar versus mesenteric, or retiform versus inosculant. The failure of PTCE was significantly higher for complex endoleaks (ie, cases where an aneurysm sac outflow vessel was angiographically visualized; 5/6, 83% versus 1/6, 17%; $P = .008$).

Table II. Endoleak characteristics and mechanisms of failure of aneurysm sac exclusion in patients with type II endoleaks treated with PTCE

Patient	*Type of endoleak treated*	*Successful sac exclusion*	*Mechanism of failure*	*Repeat PTCE*
1	Inosculant, iliolumbar	No	New mesenteric type II endoleak	Yes
2	Inosculant, mesenteric	No	Persistent flow through coils*	No
3	Retiform, iliolumbar	No	Persistent flow through coils†	Yes
4	Inosculant, iliolumbar	No	Retiform collaterals around coiled artery	No
5	Inosculant, iliolumbar	No	Retiform collaterals around coiled artery	No
6	Inosculant, iliolumbar	No	Retiform collaterals around coiled artery	No
7	Inosculant, mesenteric	Yes		
8	Inosculant, mesenteric	Yes		
9	Inosculant, iliolumbar	Yes		
10	Retiform, iliolumbar	Yes		

*Patient on chronic anticoagulation therapy for prosthetic heart valve.
†Technically inadequate PTCE.

In comparison of the preoperative and last follow-up CT scan measurements of maximum aneurysm diameter for all 14 patients with type II endoleaks (mean follow-up time, 18.2 months; range, 3 to 36 months), no significant difference in the change in aneurysm diameter after er-AAA was found between patients with persistent (n = 8) versus sealed (n = 6) endoleaks (mean change in maximum aneurysm diameter, +0.3 mm versus −1.7 mm, respectively; $P = .37$). There were no major complications or deaths after PTCE. A dissection of the internal iliac artery was the single minor complication. PTCE of the postdissection iliac artery pseudoaneurysm was subsequently successful.

DISCUSSION

Although aneurysm ruptures after er-AAA have resulted from type II endoleaks, the significance of persistent type II endoleaks remains controversial.[4-6] Some type II endoleaks have been associated with aneurysm expansion, whereas others have not.[9-12] In the Eurostar series of more than 2000 patients, a persistent type II endoleak was found to be a significant risk factor for late conversion, although not aneurysm rupture, and was associated with a combined rupture/conversion rate of 5.3%.[13,14] In animal models with type II endoleaks, systemic sac mean and pulse pressures were found to correlate with the diameter of the patent collateral vessel.[15,16] Similar results also have been reported in mechanical endoleak models.[3,17] In addition, systemic or near systemic sac pressures have been measured in patients with type II endoleaks associated with aneurysm enlargement with selective IMA catheterizations and with translumbar punctures.[2,18] We believe that the current evidence strongly supports an aggressive approach to obliterating type II endoleaks, although the long-term effectiveness of the prevailing treatments for type II endoleak remains uncertain.

Since first reported in 1997, PTCE has emerged as the main treatment of type II endoleak.[19] In most patients in whom angiography shows an inosculant collateral pathway from either a mesenteric or internal iliac artery to the aneurysm sac, microcatheter access to the sac and affluent arterial orifice can be achieved and coil deployment successfully accomplished. Despite the high initial technical success rate of PTCE, achieving complete aneurysm sac exclusion with coiling the endoleak channel is far less certain. All reports of endoleak PTCE to date have involved small numbers of patients, and the rate of successful endoleak resolution on follow-up imaging studies has varied widely from 9% to 100%.[18,20,21] In our series, initial technical success was achieved in nine of 10 patients but was successful in completely excluding the aneurysm sac in only four patients. Repeated PTCE was performed in two patients, of whom one had successful sac exclusion. Moreover, the efficacy of successful PTCE in depressurization of the excluded sac is unknown. Although a decrease in aneurysm size after successful embolization has been reported,[22,23] continued sac pressure transmitted through the thrombosed endoleak (endotension) has been inferred as the cause of continued aneurysm growth observed in some patients despite successful PTCE.[15,24] In this series, no significant differences were found in either mean AAA diameter change or the incidence of increased versus decreased AAA diameter between patients with persistent (n = 8) versus sealed (n = 6) type II endoleaks. We continue to follow all of these patients closely because the long-term effects of persistent or sealed type II endoleaks are still unknown.

Because CT scanning, duplex ultrasound scanning, and three-dimensional reconstruction imaging cannot reliably determine the type and source of an endoleak, all patients found to have endoleaks with surveillance CT scanning need angiography. Angiography allows type II endoleaks to be further subclassified by the route of collateral flow as iliolumbar or mesenteric types. Although these two collateral pathways predominate, endoleaks from patent accessory renal and middle sacral arteries have been reported.[6,25] We further categorized type II endoleaks on the basis of the character of the collateral anastomotic pathway as retiform or inosculant, which may significantly impact on the role and results of PTCE. Presuming there to be flow through an endoleak, an inosculant type II endoleak, composed of a

large dominant vessel, should theoretically transmit higher arterial pressure to the aneurysm sac and increase the likelihood of expansion or rupture compared with a retiform anastomosis. Moreover, inosculant type II endoleaks may be more effectively treated with PTCE than retiform endoleaks because selective catheterization and coil deployment of a single large collateral vessel is more easily achieved. For most of the retiform endoleaks we encountered, including four patients found initially to have retiform endoleaks and three patients in whom treatment failed by retiform development, selective catheterization and coil embolization was not considered to be technically feasible. Of the two retiform endoleaks that were treated, one resolved and one has persisted despite two PTCE procedures. Because of the small numbers of patients, we were not able to show a statistical difference in the outcome of PTCE between retiform and inosculant endoleaks. Nevertheless, we believe that the character of the collateral anastomotic pathway is important and that PTCE will rarely be an effective treatment for retiform type II endoleaks.

An emerging model of type II endoleaks distinguishes between simple endoleaks with one feeding vessel and those with multiple vessels communicating with the aneurysm sac, which may also have prognostic and treatment implication. Conventional angiography may not adequately visualize all endoleak branches and may underestimate the complex nature of the endoleak.[26,27] The inability of PTCE to reliably occlude any but the one selectively catheterized inflow vessel of a complex endoleak may explain some of our PTCE failures.

Baum et al[27] have suggested that complex endoleaks behave like arterial malformations and that translumbar embolization is the treatment of choice. They reported that direct translumbar embolization was effective in elimination of the endoleak in 92% of cases, as compared with an 80% failure rate for transluminal PTCE. In this model, transluminal embolization of a single feeding vessel is ineffective, whereas translumbar embolization of the aneurysm sac occludes the communication between the aortic side branches analogous to embolization of the central nidus of an arterial malformation and was successful in 12 of 13 patients.[27] In our series, visualization of an outflow vessel was predictive of PTCE failure. Because visualization of an outflow vessel is indicative of a complex endoleak, this model would explain our failures in this subset of patients.

Other techniques for treatment of type II endoleaks are currently being investigated. Transluminal injections of hystoacryl glue[23] and ethylene-vinyl-alcohol copolymer,[28] instead of coils, have been reported with success rates of 89% and 83%, respectively. However, complications of transluminal injection of liquid agents have been observed, including colonic ischemia and paraplegia.[12,29] Successful obliteration of a type II endoleak with laparoscopic clipping of the offending collateral vessels has also been reported, but morbidity and failure rates are unknown.[30] Quite promising are the reports by Walker, Macierewicz, and Hopkinson[31] of prevention of type II endoleaks with intraoperative packing of the aneurysm sac with collagen sponge at the time of endografting. This technique reliably achieves complete aneurysm sac thrombosis, even in patients with numerous patent aortic side branches, drastically reducing rate the incidence of type II endoleak.[32]

Currently the natural history and best treatment of type II endoleaks are unknown. Although the number of patients reported herein was too small to make meaningful comparisons between groups, the multiple mechanisms by which endoleaks persisted after PTCE (Table II) suggest that this treatment is inadequate and more than one method of treatment may be needed to achieve aneurysm sac exclusion. Continued experience may allow tailoring of the treatment of type II endoleak to best match the anatomic environment. Perhaps retiform pathways will be more effectively obliterated with use of liquid agents. Endoleaks with visible outflow vessels might respond better to a translumbar approach by which both inflow and outflow vessels can be addressed. Patients who need chronic anticoagulation therapy may benefit from intraoperative packing. Regardless of future advances in the treatment of type II endoleaks, close monitoring and serious consideration of conversion to open repair for aneurysm enlargement will continue to be necessary to assure superior long-term results of er-AAA.

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DISCUSSION

Dr Alan Lumsden (Houston, Tex). Dr Solis and his colleagues examined the efficacy of coil embolization on the sealing of endoleaks with defined type 2 anatomy. They elected to treat all endoleaks regardless of aneurysm status. Most of these aneurysms were stable in size. We recently reported an intervention on a patient with a stable aneurysm where we performed IMA embolization complicated by colon ischemia and have completely changed our approach to the presence of an asymptomatic endoleak in a stable aneurysm.

The Southern Illinois group had a surprisingly low type 2 endoleak; their overall endoleak was 14.6%, but only approximately half of these actually had type 2 endoleaks. Of the 28 patients who they identified with endoleaks, only 23 of 28 underwent angiography, of which 14 had defined type 2 endoleak anatomy. Angiography was performed in all of these patients with an intent to treat with embolization in those who were amenable to embolization. However, basically it was performed in only 10 of the 14 patients because it was deemed to be technically not feasible in four out of the 14; in other words, a 28% up-front intention to treat failure rate. This was because of the presence of retiform iliolumbar-type anatomy. The study really then examines the remaining 10 patients who were treated with embolization. However, a recurrent, or was it persistent, endoleak was found in nearly 60% of these cases. The failure modes of this embolization were threefold: number one, appearance of new endoleaks through new lumbar arteries, which were not previously visualized. In one case, there was a persistent flow through coils, albeit in an anticoagulated patient. And the third mechanism was that there were new collaterals or, as they define it, retiform collaterals around a successfully occluded artery. Each of these failure modes, of course, has a different mechanism and begs several questions.

Most surgeons do not have a lot of experience with embolization, and it is difficult to define what the end point is. When did you determine that there were enough coils placed to successfully achieve occlusion of that artery? How do you deem when the embolization procedure is adequate?

The second question is given the very high likelihood that the interventions are either not feasible or associated with failure, ultimately this has not helped most of the patients that you performed angiography on. Has this subsequently changed your selection criteria for intervening on a patient with a demonstrated type 2 endoleak and a nonexpanding aneurysm?

Question number three is, how aggressive are you in injecting the sac to visualize outflow vessels, and do you think failure to identify concurrent outflow vessels may have contributed to the subsequent failures?

A final question is clearly there are many approaches to performing embolization. Could you discuss the role for other agents,

such as thrombin injection, onyx, glue, or have you changed a direct sac puncture?

My final question is do you think there is a role for laparoscopic ligation of the inferior mesenteric artery? That has become a preferred method of intervening when a type 2 endoleak anatomy is based upon reflux in IMA.

Thank you for the privilege of discussing this paper.

Dr Maurice M. Solis. Thank you very much, Alan. Regarding the question of how many coils we put in and when do we know that we have adequately occluded the vessel, it is completely subjective. We essentially keep deploying coils until there appears to be a large collection of coils in the leak. There is absolutely no science behind that. In fact, we do not even use the presence of continued flow through the coils as an endpoint because generally these patients are anticoagulated during the procedure. We commonly do not see the vessel completely during the procedure.

Has our high failure rate changed our thoughts on intervening on these patients? No. We still feel that the current evidence suggests that a type 2 endoleak is a pressurized sac and is going to be associated with a poor prognosis and aneurysm expansion, and we do not really wait to see aneurysm expansion. We try to treat these aggressively. However, it is changing how we intervene on these patients because we are going to translumbar puncture, and we are looking at the other agents, although we have no experience with tissue glues, or with thrombin injections. Your experience with the thrombin injections has actually tempered our enthusiasm for that technique.

In regards to laparoscopic clipping of the IMA, in our experience the IMA seems to be the easiest and most reliable to embolize and so we are going to continue embolizing the IMA endoleaks. I think coil embolization is more reliable for mesenteric endoleaks that are always inosculant via a large marginal artery collateral.

Dr Frank Veith (Bronx, NY). I enjoyed this paper. We have had a long interest in type 2 endoleaks and regard them as the Achilles heel of endografting. As you know, some people think that type 2 endoleaks are of no consequence. You obviously are very aggressive and treat all or almost all of them. We are somewhere in between. We think that a type 2 endoleak associated with a stable or shrinking aneurysm probably does not need to be treated.

We have had 16 patients with persistent type 2 endoleaks, and in 6 of those, there has been AAA enlargement, while in the others the AAAs are stable or shrinking. Like you, we started out with transarterial embolization and were very aggressive about it. However, it often does not work for the reasons that you put forth. We consider type 2 endoleaks either complex with multiple feeding vessels or simple with to and fro input. It is the complex ones that cannot always be treated by transarterial embolization, at least in our experience. So we have gone to translumbar techniques, and some of these cases have been extraordinarily gratifying when we have actually been able to put the catheter into the IMA, into the lumbars, and embolize them directly. However, sometimes one cannot do that and then one puts the coils or glue right into what I call the engine room on the nidus. In many cases, this has resulted in resolution of the endoleak, but we have also occasionally been frustrated with translumbar techniques. Occasionally we cannot get into the engine room, we certainly cannot always get into the branches, and some of the patients' leaks have persisted. I have a couple of questions for you.

One of the question is, how many or what percentage of these type 2 endoleaks are associated with aneurysm enlargement? And rupture? I have tried over the years to collect ruptures. You mentioned in one of your slides ruptures. I have six cases, including one of our own, where rupture has occurred. However, as you know, those who say these are benign, they say, oh, well, they never rupture. Well, they certainly do, and I would be interested in knowing what percentage of type II leaks do you think enlarge and what is the incidence of rupture? How many cases have you been able to collect with rupture?

Dr Solis. We have not seen any ruptures. Regarding the number of patients that have enlarged of the 14 patients that had type 2 endoleaks, we ended up with six sealed endoleaks and the others have persistent endoleaks, either because they were not treated or they were treated and failed. In the sealed group, one has had a small, probably insignificant increase. In the persistent group, one has had significant increase and underwent translumbar embolization last week. Two have had minor, 2 or 3 mm, increases that we are watching. So, aneurysm increase regardless of whether the endoleak has persisted or sealed has occurred in the minority of cases.